

of people affected are likely to increase as the modern world places increasing demands on the human voice via mobile telephones, voice recognition software, and interpersonal verbal communications. In advanced societies, the voice is essential for approximately one third of the labour force.⁵

However, change in the quality of the voice may also be the first sign of a wide variety of systemic, neurological, and structural disorders.²⁻⁶ Early and subtle laryngeal manifestations may be the result of a variety of systemic illnesses including bacterial, viral, and fungal infection, rheumatoid arthritis, hypothyroidism, gastro-oesophageal reflux, and vascular and cardiac disease.²⁻⁶ Isolated change in the voice may also indicate early lower motor neurone disease (for example, Parkinson's disease, myasthenia gravis, or benign hereditary "essential" tremor) or focal upper motor neurone lesions.²⁻⁶ Structural lesions on the vibrating edge of the vocal folds affect their mass, regularity of vibration, and adduction. These structural changes can be the result of many factors including voice abuse (vocal nodules, vocal fold haemorrhage), viruses (laryngeal papillomatosis), inflammatory irritants (Reinke's oedema, contact granulomas), congenital malformation (laryngeal web, laryngomalacia), and malignancy.^{2-6,7}

The science of diagnostic laryngeal or voice pathology has been revolutionised in the past 20 years.²⁻⁸ Technological advances in laryngeal examination and vocal function have led to a more precise understanding of the function and dysfunction of the vocal tract.⁷⁻⁸ Fibreoptic endoscopy enables extensive examination of the laryngeal structures during all types of vocal activity (for example, speaking, singing, and shouting). Applying the principles of stroboscopy (a means of "sampling" images to create the visual illusion of "slowing down" the movement) to the endoscopic examination allows detailed observation of the membranous layer of the vocal folds during phonation.²⁻⁷ Voice pathologists use other instrumental and perceptual techniques to measure a wide range of aspects of voice production including vocal frequency, amplitude, stability, range, regularity, and aerodynamic efficiency.⁸

These advances in voice clinics and voice laboratories have resulted in greater accuracy of diagnosis and better selection of treatment.^{2-7,8} Appreciation of the subtle vibratory (mucosal waveform) characteristics of the vocal folds has dramatically changed the principles of surgical intervention for pathological laryngeal disorders.^{2,7} Microlaryngeal surgery now

extends beyond the primary aim of establishing a histological diagnosis and incorporates a wide variety of techniques to restore or improve the disordered voice.²⁻⁷ Voice therapy remains the treatment of choice for most non-organic and some organic voice pathology.⁸⁻⁹ One achievement of recent research in voice pathology has been the establishment of level II evidence to support the efficacy of voice therapy for the most common disorders.¹⁻⁹

Despite all of these advances, clinical research in voice pathology still remains in its infancy. The tools of diagnosis and functional measurement are now ready for rigorous application to the clinical field.⁸ For example, the impact of gastro-oesophageal reflux disease, inhalatory steroids, and mental illness on voice disorders are largely unknown.²⁻⁶ Equally, the techniques of differential diagnosis for less common disorders such as laryngeal dystonia, superior laryngeal nerve paralysis, and organic tremor remain controversial.²⁻⁶ Furthermore, the pathophysiology of laryngeal disorders such as papillomatosis, polypoidal degeneration, and paradoxical vocal fold movement are poorly understood.²⁻⁶ The efficacy of surgical techniques to improve voice quality (phonosurgery) remains largely unevaluated.⁸ Clinical research needs to complement the growth in clinical services. Several postgraduate specialist research degrees now exist, and some doctoral students with a clinical background are being attracted into the field. Hopefully with an appropriate academic infrastructure in place these issues in research in voice pathology will begin to be addressed.

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Social anxiety disorder

Is common, underdiagnosed, impairing, and treatable

The hallmark of social anxiety disorder is extreme and persistent fear of embarrassment and humiliation.¹ People with this condition (which is also known as social phobia) often avoid participating in social and public activities, such as public speaking, social gatherings, or meetings. Whereas normative social anxiety may serve to focus attention and prevent inappropriate behaviour, the intense symp-

toms of social anxiety disorder, by definition, interfere with functioning or cause marked distress.

Differentiation of social anxiety disorder from other phobic disorders was first validated by its characteristic age of onset in the mid-teens and greater ratio of men to women.² Although once believed to be uncommon, social anxiety disorder was found to be the third most prevalent psychiatric disorder in the US

national comorbidity survey,³ and most studies in Europe and North America have found a 7-12% lifetime prevalence in the community and higher rates in primary care samples.⁴

Despite increasing recognition of social anxiety disorder as common, impairing, yet treatable, it often remains undiagnosed.⁴ The reticence and shame that are intrinsic to social anxiety disorder inhibit help seeking. Persons usually come for treatment only after years of suffering and may present initially with a complication, such as major depression or alcohol abuse. Clinicians can encourage patients to discuss their symptoms by including a brief query into social anxiety or avoidance in a review of systems assessment.

In feared situations, patients with social anxiety disorder typically experience self consciousness, embarrassment, and difficulty speaking. Autonomic arousal symptoms, such as blushing, sweating, trembling, and palpitations, are sometimes prominent. Thoughts often dwell on inferiority to others, desire to flee, and anticipated negative evaluation by others. Weeks of anticipatory anxiety may precede a social event, with self deprecating thoughts and demoralisation in its aftermath.

Persons with the generalised subtype of social anxiety disorder, defined by fear of most social situations,¹ are most impaired, most likely to seek treatment, and have been studied most in clinical trials.⁵ Patients with the non-generalised form are typically comfortable in informal social settings but experience distressing or impairing anxiety attacks during public speaking or performance.¹ The anxiety related symptoms of social anxiety disorder may take the form of a panic attack, but they can be differentiated from symptoms of panic disorder by their consistent relation to social cues. With its trait-like early onset, pervasiveness, and high chronicity, social anxiety disorder may be alternatively conceptualised as the pathologically extreme form of the continuum of social anxiety present in the community.

The best established treatments for social anxiety disorder are cognitive behaviour therapy and serotonin reuptake inhibitors. Cognitive behaviour therapy addresses a dysfunctional cycle in which avoidance of feared situations diminishes opportunities for social growth and reinforces fears, which in turn lead to further avoidance. The therapist works in the here and now to help the patient identify fears and avoidance, develop more productive coping thoughts and behaviours, and systematically confront feared situations while using new coping techniques. Controlled studies show clinical improvement over 12-16 weekly sessions,⁶ with gains usually persisting after discontinuation of therapy.⁷

Selective serotonin reuptake inhibitors are the best established drug treatment for social anxiety disorder, with controlled trials showing efficacy for paroxetine, sertraline, and fluvoxamine.⁸ Doses typically used for depression seem adequate, but many patients will require a longer 12 week trial to establish clear outcome. Drug treatment should continue for 6-12 months after response, and some patients may require longer treatment to maintain gains.⁷ Alternative drugs include the monoamine oxidase inhibitor phenelzine, which has been shown to be effective in several controlled trials but is usually reserved for patients who do not respond to

other treatment. This is due to the need for dietary restrictions and risk of hypertensive reaction.⁶ The newer reversible monoamine oxidase inhibitor moclobemide has seemed effective for social anxiety disorder in some but not all controlled trials.⁹ The benzodiazepines clonazepam and bromazepam and novel anticonvulsant gabapentin have been shown to be efficacious in single controlled trials. β adrenergic blockers have not differed from placebo in efficacy for treatment of generalised social anxiety disorder, but they seem to be useful taken on an as-needed basis in treatment of the non-generalised subtype. No Cochrane reviews of social anxiety disorder have been undertaken.

Twin and family studies show substantial genetic and environmental contributions to social anxiety disorder.¹⁰ Studies of toddlers exposed to novel stimuli have suggested that behavioural inhibition coupled with autonomic reactivity characterise a relatively stable temperament that predicts development of social anxiety in adolescence.¹¹ Parenting that is overprotective yet critical may contribute to the development of social anxiety disorder in vulnerable children. Several neurotransmitter systems, including dopamine and serotonin, manifest differences in social anxiety disorder, and subcortical fear circuitry involving activation of the amygdala may be hypersensitive and easily conditioned to social stimuli.¹²

As social anxiety disorder has gained recognition as a common, impairing, and treatable condition, more attention has focused on improving detection of serious cases in the community. Earlier identification of symptomatic individuals holds promise for prevention of long term dysfunction and complications.

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